Helminths

Introduction

This is the lowest-yield lesson in the entire Microbiology module. We begin with an appeal to have you skip this lesson. There are more helminths you need to know than there were protozoa. Treatments for helminths have more bizarre names (pick albendazole). The life cycles of helminths are more complicated, and there are so many overlapping similarities in the syndromes and pathogenesis of disease, it is really easy to spend a lot of time studying helminths only to end up confused. We go over these worms the best we can. We also provide a medium-yield table at the end, and a highest-yield-you're-going-to-get-from-worms table after that. If you insist on starting down the path of studying helminths, brute-force memorization IS a tactic we suggest you employ for your exam. Like the protozoan lecture, if you are studying tropical medicine, please do not be offended. HUMAN LIFE CYCLES ONLY. Dr. Williams did not study helminths at all for his Step 1 exam and he did pretty darn well.

Helminths are **multicellular organisms**; they have anatomical sex, they mate, and they reproduce. They are worms. Helminths have three forms. They start as an **egg**, which contains the embryo. The egg hatches and differentiates into a **larva**. The larva then matures into the **adult** form, which can mate, producing more eggs. The maturation from larva to adult is considered sexual development. Sexual development occurs in the **definitive host**. It is where the adult lives and lays eggs. The **intermediate host** is where the eggs turn into larva. Humans can be definitive hosts, intermediate hosts, or **dead-end hosts** where the larval forms in the human cause that human to have a disease, but the larvae are not transmitted to other humans or animals from the human.

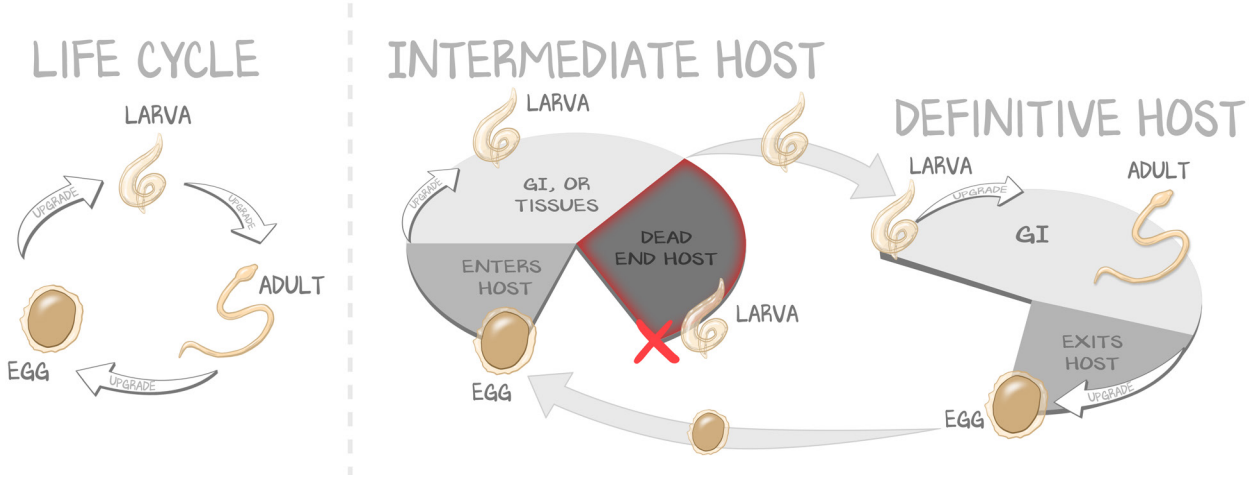

Figure 2.1: Helminth Life Cycle and Host Types
The general life cycle of all helminths is between three stages. Adults reproduce sexually and lay eggs. Eggs germinate into larvae. Larvae grow up to be adults. Certain events tend to trigger an upgrade, a passage to the next stage of the life cycle, such as ingestion. Sometimes it is just the passage of time. Regardless, an egg becomes a larva, a larva becomes an adult, and adults lay eggs. Throughout that life cycle, the helminth may spend time in different hosts. Some helminths pass through all stages in one organism. Others go between species. When between species, the definitive host is the host in which the adult reproduces and lays eggs. The intermediate host is where the egg turns into the larva. That intermediate host is meant to be eaten by the definitive host, taking the larva into the definitive host's gut to turn into an adult and reproduce. Sometimes the intermediate host is a human, who tends not to be eaten by other animals. When the intermediate larva ends up in a human, where it will not be taken into the definitive host, the human is termed a dead-end host.

INTESTINAL NEMATODES	TISSUE NEMATODES	CESTODES	TREMATODES
Enterobius	*Onchocerca*	*Taenia solium*	*Schistosoma*
Ascaris	*Loa loa*	*Diphyllobothrium*	*Clonorchis*
Strongyloides	*Wuchereria*	*Echinococcus*	
Ancylostoma, Necator			
Trichinella			
Toxocara			

Eat eggs or larvae	Insect vector		Snails and fish

Table 2.1: Worms

Intestinal Nematodes

Intestinal nematodes live in the human intestines. How they get there is variable, and ends up being quite a convoluted tale. In every case the worm burrows through something, gets into the blood, and goes somewhere else, usually the lungs, to be coughed up and swallowed again. Because the worm has to burrow through something—skin, intestinal mucosa, whatever—and because they are so large (multicellular organisms), they provoke a systemic **eosinophilia**. For those that go through skin, "skinflammation" will result. For those that go into the lung, pulmonary eosinophilia is a possibility. While there are many commonalities, use caution: no two mechanisms are the same. But one feature is true for all intestinal organisms. Every time a worm is swallowed, no matter by what organism, it gets an "upgrade." Being swallowed advances the life cycle one stage—egg to larva, larva to adult.

***Enterobius* = Pinworm.** Male and female worms live in the intestinal tract (cecum). The male fertilizes the female's eggs. The female migrates to the perianal region, deposits those eggs at the anus, and then swims back to her papi in the cecum. Human babies, who put their hands everywhere, including their mouth, get an itchy butt from the eggs. Baby scratches butt. Baby inserts eggs into mouth. Baby itches butt. Baby touches everything, placing eggs everywhere. Any human who then ingests the thing baby touched with eggs, gets egged. Eggs are already fertilized. Larvae hatch in the intestines, where they penetrate the mucosa to develop. Once developed, they come back out of the mucosa to join the fam in the colon. They DO NOT go through the mucosa, only into it. So . . . minimal inflammation and only local. NO systemic inflammation, NO eosinophilia. The **female migration to perianal skin occurs when it is dark**, usually at night. The **adhesive tape test** (Scotch tape test) is performed before baby has a chance to poop in the morning. A piece of adhesive tape is placed over the anus to capture the eggs meant for baby's fingers. Eggs and worms are NOT found in stool. Treatment is with albendazole.

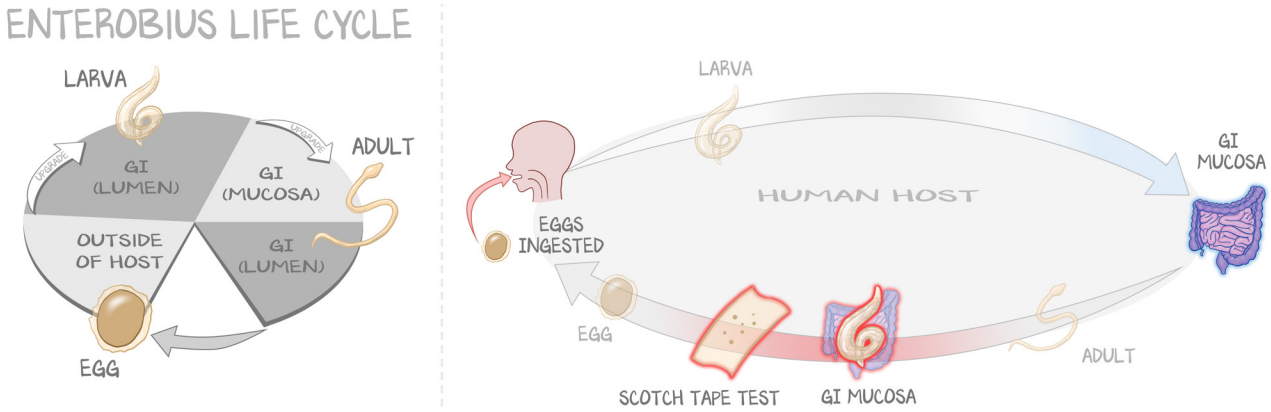

Figure 2.2: *Enterobius* = Pinworm
Start with the human head and follow the arrows with the text. The human swallows an egg, where the egg upgrades to a larva in the gut. It burrows into, but not through, the GI mucosa and becomes an adult. Then it travels to the colon to mate with other pinworms, laying eggs. Eggs are deposited on the anus to be scratched, taken up on fingers, and ingested, where the cycle begins anew. The only consequence is an itchy butt and the ability to dab the anus with adhesive tape to diagnose the condition. The life-cycle pie in general is on the left; the details and consequences of the life cycle are on the right. All life-cycle maps will be from the perspective of the human.

***Ascaris* = Giant Roundworm.** Male and female worms live in the intestinal tract (small intestine). Eggs are fertilized and released into stool. Fecal-oral route, eggs are ingested in the mouth. Eggs are swallowed, so they UPGRADE. Eggs hatch larvae in the small intestine. Those larvae don't just get into the mucosa to mature; they **burrow THROUGH the gut wall into the bloodstream**. They migrate throughout the bloodstream and come out in the **lungs**, where they migrate out of the blood into alveoli. Worms in alveoli are irritating to the alveoli. The person coughs. Larvae are passed up trachea into oropharynx. Many larvae are expelled. But those that don't quite make it out of the mouth? Human swallows. UPGRADE! Now the larvae mature into adults. Because the larvae are foreign organisms and have foreign antigens, when they hang out in the alveoli waiting to be coughed up, they induce an antigenic response called **pulmonary eosinophilia** (the eponym is Löffler syndrome). These are worms, multicellular organisms, far larger than a neutrophil or macrophage. Phagocytosis isn't going to work here, which is why eosinophils predominates. The immune response to large organisms is to flush them out using IgE, mast-cell degranulation, and a little help from eosinophils. The patient does still have an inflammatory response that looks like a **pneumonia**—fever, cough, dyspnea, consolidation on X-ray—with a **parapneumonic effusion**—eosinophilic exudate. Sputum samples will reveal eosinophil-derived Charcot-Leyden crystals. Because a large organism is going through tissues, **systemic eosinophilia** will be found on labs. If the worms are not taken care of, they will grow, continuing to eat what the stomach and duodenum digest, which can result in **malnutrition**. If they get even bigger, they can cause **intestinal obstruction**. These worms' eggs are visible **in stool**, which will show a **scalloped edge**. *Ascaris* does not attach to the gut wall; it swims in the fluid in the intestine. Treatment is with albendazole or ivermectin.

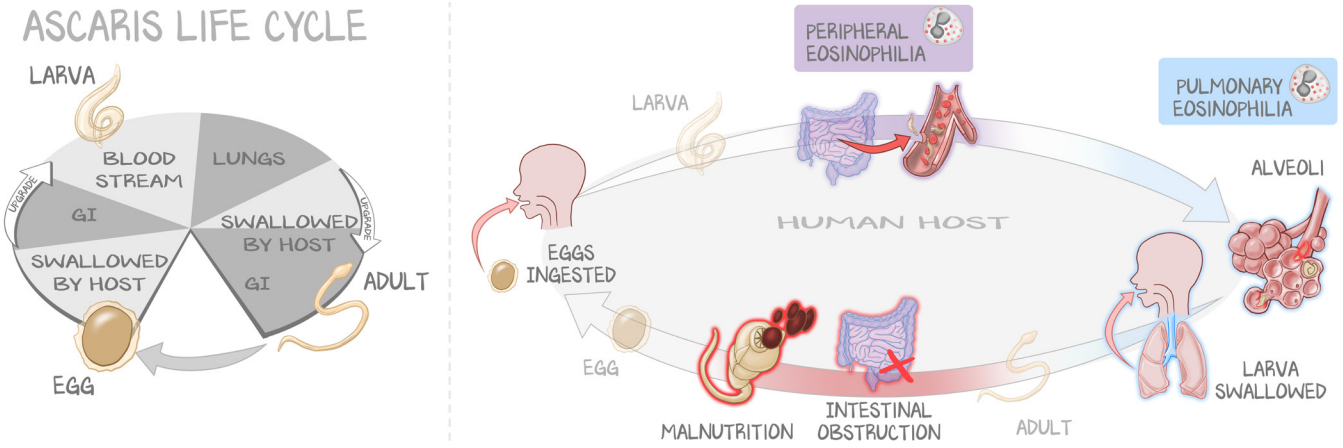

Figure 2.3: *Ascaris*
Start with the human head and follow the arrows with the text. The human swallows eggs, where the eggs upgrade to larvae in the gut. The larvae penetrate the GI mucosa and enter the bloodstream provoking peripheral eosinophilia. They swim to the lungs, where they cause pulmonary eosinophilia. They are coughed up, then swallowed to become the adult. The adult eats the human's food, causing malnutrition, and grows large, causing intestinal obstruction.

***Ancylostoma, Necator* = Hookworm.** The life cycle of hookworm is the same as roundworm: larvae get to lungs through the bloodstream, are coughed up, then swallowed . . . UPGRADE! and mature into adults in the small intestine, where they lay eggs that are passed into the stool. The stool goes into the ground. The eggs hatch in the soil. Human not wearing shoes steps on soil with feces in it. The **filariform larvae** (the infectious but nonfeeding form) get into the body **through the skin** (not eggs through the mouth like *Ascaris*; larvae go through the skin in *Ancylostoma*). Once through the skin, they ride the bloodstream to the lungs. Going through the skin is like *Ascaris* going through the GI mucosa, provoking systemic eosinophilia. Going to the lung to be coughed up and swallowed like *Ascaris* means that pulmonary eosinophilia is possible with this worm as well. What is different between the worms is how they get in and what the adults do in the intestine. *Ancylostoma* adults in the small intestine attach to the gut wall using cutting plates or teeth, and **drain blood**. Because they are not eating the food humans digest, there is NO malabsorption. Because they are leeching blood, there can be instead a **microcytic anemia**. Larvae penetrate the skin, resulting in **cutaneous larva migrans**, the rash (cutaneous) caused by the larvae (larva) moving through the skin (migrans). Cutaneous larva migrans is an intensely pruritic, serpiginous rash on the skin (usually foot) where the entry occurred. See the **eggs in stool**. Larvae hatch and grow in soil. The eggs don't need to be swallowed to get their life-stage upgrade. Treat with albendazole or pyrantel pamoate.

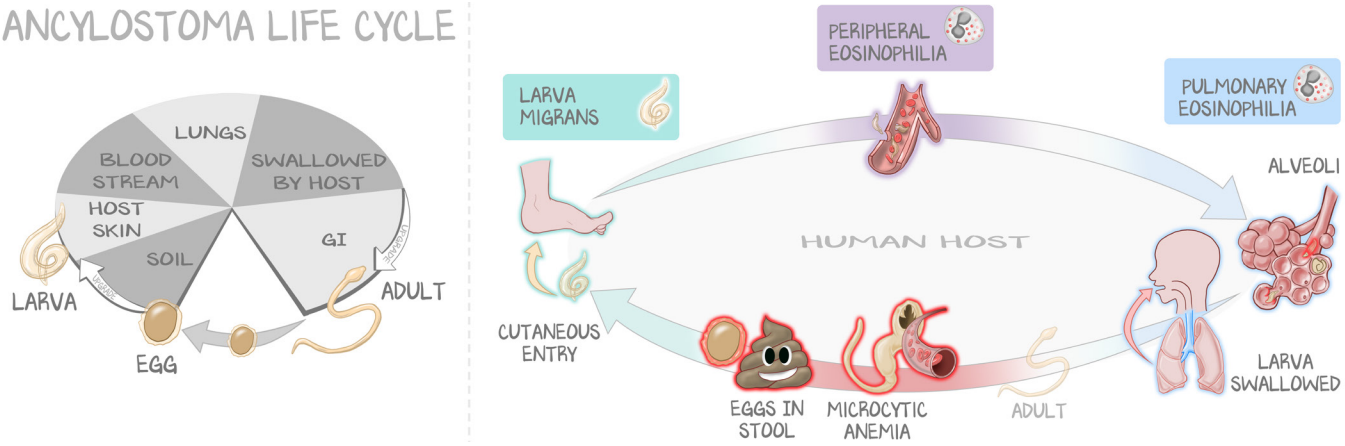

Figure 2.4: *Ancylostoma* = Hookworm
Start with the human foot and follow the arrows with the text. Larvae in soil penetrate the human foot, inducing cutaneous larva migrans. The larvae get into the bloodstream, provoking peripheral eosinophilia. They swim to the lungs, where they cause pulmonary eosinophilia. They are coughed up, then swallowed to become the adult. The adult steals the human blood, causing a microcytic anemia. All the while, it is laying eggs the human poops out. Being in the soil is enough for the upgrade to the larvae.

***Strongyloides* = Threadworm.** Filariform larvae penetrate the skin of the patient, get into blood, go to lungs, get coughed up, and are swallowed, where they mature into adults. Löffler syndrome, cutaneous larva migrans, systemic eosinophilia: all part of this syndrome for the same reason they were part of the previous syndromes. All of the worms so far have passed fertilized eggs into the stool. However, *Strongyloides*' **eggs** are laid AND **hatch in the intestinal mucosa**. It is the **larvae** that are passed in stool. The larvae can be passed in stool OR the larvae can just go through the wall of the intestines without being passed. This hatching and infecting is called **autoinfection**. Autoinfection comes only with **intense immunocompromise**. The worms that hatch and bore have a higher likelihood of burrowing more distal in the intestines (laying, hatching, and turning to a larva takes time), including the colon. Translocation of worms through the mucosal barrier into the bloodstream can lead to translocation of Gram-negative rods and anaerobes from the colon into the blood, resulting in Gram-negative rod sepsis. Because the eggs hatch in the intestine, you will want to look for **larvae** in the stool.

If eggs are ingested (not the usual way), UPGRADE, larvae form in intestine, burrow, and cause disease. If larvae get into the skin, they go to lungs, cough, swallow, UPGRADE, adults lay eggs. Those eggs can either become larvae in stool (no disease, immunocompetent), or those eggs can become larvae in the intestines and burrow right away (autoinfection, immunocompromise, both systemic *Strongyloides* AND Gram-negative rod bacteremia).

Treatment is with ivermectin or albendazole.

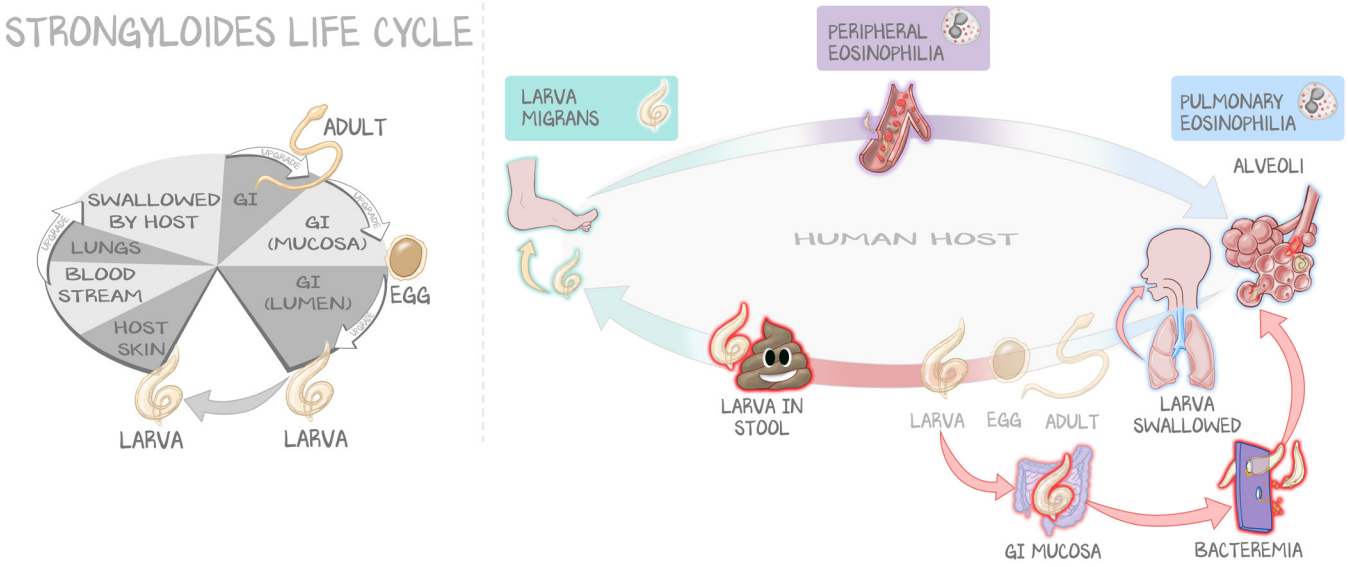

Figure 2.5: *Strongyloides* = Threadworm
Start with the human foot and follow the arrows with the text. Larvae in soil penetrate the human foot, inducing cutaneous larva migrans. The larvae get into the bloodstream, provoking peripheral eosinophilia. They swim to the lungs, where they cause pulmonary eosinophilia. They are coughed up, then swallowed to become adults. The adults lay eggs, which become larvae in stool to repeat the process. Normally, eggs are released into stool, where they become larvae by the passage of time. But these larvae can burrow through the GI mucosa into blood. Previous GI-burrowers did so in the small intestine, which is sterile. Sometimes, especially with a high disease burden, Strongyloides lays eggs soon enough that they hatch in the human and penetrate the GI mucosa. But because the larvae first upgrade to adults, then lay eggs, the helminth becomes the GI-mucosa-penetrating larva in the colon, which is not sterile. The penetration of the GI mucosa by the helminth can bring bacteria with it.

***Trichinella* = cysts in skeletal muscle.** *Trichinella* hides in cysts in skeletal muscle of animals, mainly pork and bear. What we call "meat" is "animal skeletal muscle." The **encysted larvae** are easily destroyed by cooking the meat. Undercooked pork or bear puts the patient at risk for ingesting the encysted larvae. In the small intestine, they come out of their cyst (excyst), mature into adults, and lay eggs in the mucosa. The larvae hatch, burrow through the mucosa into the bloodstream, and go to **skeletal muscle**, where they encyst again. Humans are at the top of the food chain. No one is going to eat the encysted *Trichinella* in human skeletal muscle, so humans become a **dead-end host** with encysted LARVAE in skeletal muscle. They will never mature, and nothing generally eats a human's skeletal muscle. A worm is going through the mucosa, so you are going to get eosinophilia. But this disease has no pulmonary or skin findings. The worms go to skeletal muscle and encyst. The encysted larvae cause **skeletal muscle problems**—myalgias, weakness, and an elevated creatine kinase. Muscle cells are dying, being eaten to make room for the encysted larvae. A biopsy is made of the skeletal muscle after other causes of weakness or myositis have been ruled out. A biopsy reveals the encysted larvae within skeletal muscle. Steroids reduce myositis; albendazole will treat the worm.

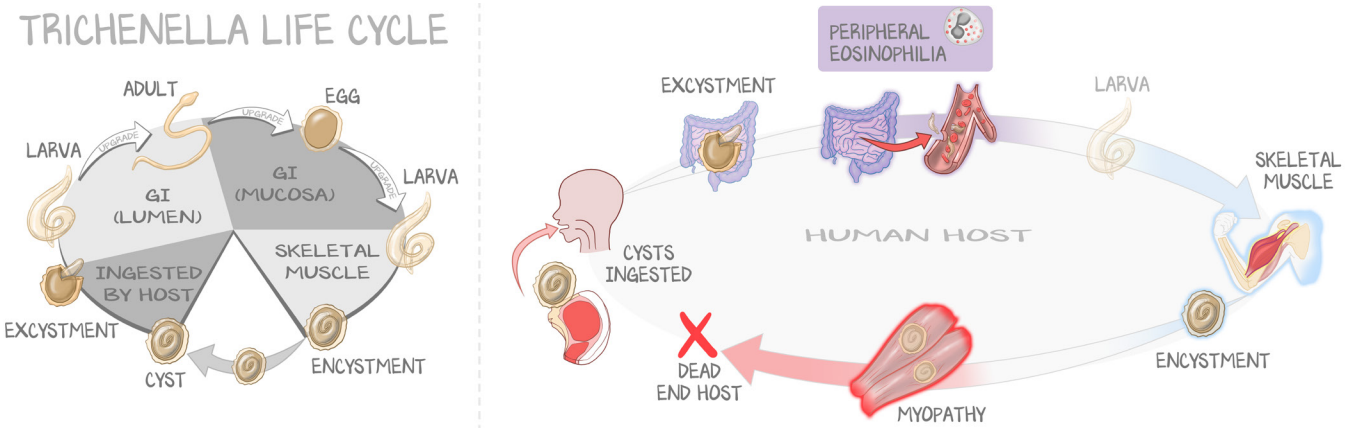

Figure 2.6: *Trichinella*
Start with the human head and follow the arrows with the text. The human eats skeletal muscle (meat) with encysted larvae, where the encysted larvae (the "eggs") upgrade to larvae. The larvae penetrate the GI mucosa and enter the bloodstream, provoking peripheral eosinophilia. They swim to skeletal muscle, where they encyst. Cysts cause damage to skeletal muscle, which hurts and causes weakness. Human meat (skeletal muscle) is not often the meal of the intended definitive host. Thus, the human is the intermediate host and the dead-end host.

***Toxocara canis*.** Dogs are the definitive host; humans are accidental dead-end hosts. The eggs are passed in dog feces. Fecal-oral route. Eggs hatch into larvae in the small intestine. They penetrate the mucosa, get into the bloodstream. Eosinophilia. These larvae go everywhere—**liver, brain, eyes, heart**—and are eventually encapsulated and die. Even though they die, they still cause disease, because encysted larvae eat the tissue they are in to make room. This can result in any organ being affected, so presents with a variety of hepatomegaly, blindness, seizures, coma, and myocarditis. If a tissue is biopsied, the larvae will be seen. This disease is very similar to *Trichinella*, except humans must eat dog-feces-contaminated water or food instead of stepping on human-feces-contaminated soil. The diagnosis is usually made with serologies; treat with albendazole.

Tissue Nematodes

These are nematodes that gain entry through the skin introduced by an insect vector. They are rare in the US. Just as the tissue and blood protozoa do not have a cyst form, tissue nematodes do not have an egg form. Instead, they have an offspring form, called microfilariae, that are carried safely within an insect vector who deposits the microfilariae into the human. They then swim through the blood until they get to the tissue they prefer. That causes disease. The mature, adult version is unfortunately called a larva in these organisms. We're going to use "microfilaria" for the offspring and "adult" for the adult form, so as to not confuse the nomenclature of what a "larva" is from the previous worms above.

***Onchocerca*.** This is an insect-vector disease. The **blackfly** bite deposits infective microfilariae into the **subcutaneous tissue** where the microfilariae differentiate into adults. This causes **dermal nodules**—little bumps where the fly bit. *Onchocerca* adults produce microfilariae (not eggs). Microfilariae are taken up on the next feed by the blackfly where they mature in the blackfly and migrate from the site of inoculation to whichever tissue they have tropism for. Microfilariae can concentrate in the eye, which is known as **river blindness**—the worm-looking thing in the eye. When it penetrates the globe it is small. When it causes blindness, it is big enough to see with the naked eye, and too big to get out of the globe. To diagnose, you want to catch microfilariae, not the adult forms. Since the nodules have the adults in them, you'll want to do the biopsy somewhere else. Treatment is with ivermectin.

Loa loa. This is another insect-vector worm disease that causes worms in the eye. Transmitted by a fly (deer fly, horse fly, mango fly) this results in an **adult worm crawling across conjunctiva**. Treat with diethylcarbamazine.

Wuchereria. A mosquito deposits larvae on skin. The larvae burrow into skin. The larvae mature into adults **in lymphatics**. The microfilariae circulate at night. The symptoms occur **9–12 months after the bite**. Adult worms **obstruct lymphatics**, causing lymphadenopathy. The lower extremity is commonly involved. Microfilariae are visualized on **blood smear** (especially at night). Because these can go anywhere (to be picked up by the mosquito), there can be Löffler syndrome, eosinophilia, etc. Treat with diethylcarbamazine. Know that this is an infectious cause of **elephantiasis**.

ONCHOCERCA	LOA LOA	WUCHERERIA
Blackfly vector	Mango fly vector	Mosquito vector
Nodules at bite site	Worms crawl over eyes	Live in lymphatics
Microfilariae in eye = blind	Diethylcarbamazine	Circulate at night
Ivermectin		Elephantiasis
		Diethylcarbamazine

Table 2.2: Comparing the Insect Vector Nematodes

Cestodes

Cestodes are tapeworms. They are intestinal diseases. Humans are the definitive host. The intermediate hosts (which house the larvae) are some form of animal. Regular old tapeworm is caused by ingesting pork or beef; *Diphyllobothrium* comes by ingesting fish; and *Echinococcus* is the exception, caused by dogs eating sheep with larvae and pooping the eggs into the water we drink.

Tapeworm = Taeniasis = *Taenia saginata*. Adults live in human intestines; adults lay eggs. The eggs are passed in the feces, which is then ingested by livestock. The larvae form muscle cysticerci in animals. Humans then ingest undercooked meat (beef, pork), ingesting the larvae into their intestine. Larvae get upgraded to adult tapeworms. Tapeworms eat the food we digest, resulting in **weight loss** and **malabsorption**. Look in the stool for the eggs or segments of the worm in stool.

Treatment is with praziquantel.

If an animal ingests the eggs, UPGRADE, that animal gets larval cysticerci in the skeletal muscle.

If a human ingests the larvae from cysticerci in undercooked meat, UPGRADE, the human gets adult tapeworm.

If a human ingests the eggs, UPGRADE, the human gets larval cysticerci in the brain.

***Taenia solium* = Cysticercosis.** If a **human eats eggs,** UPGRADE, larvae form cysticerci in humans, which can be anywhere, but you should learn it as the brain. In **neurocysticercosis** the lesions are in the brain, visible **cystic CNS lesions** presenting with **seizures** and **stroke-like symptoms**. A diagnosis is confirmed by a biopsy, but brain biopsies are not easy to do and are high-risk, so imaging usually will suffice. Neurocysticercosis is treated with steroids to reduce inflammation, albendazole to kill the worm, and antiepileptics for seizure prophylaxis. Non-neurocysticercosis is treated with praziquantel.

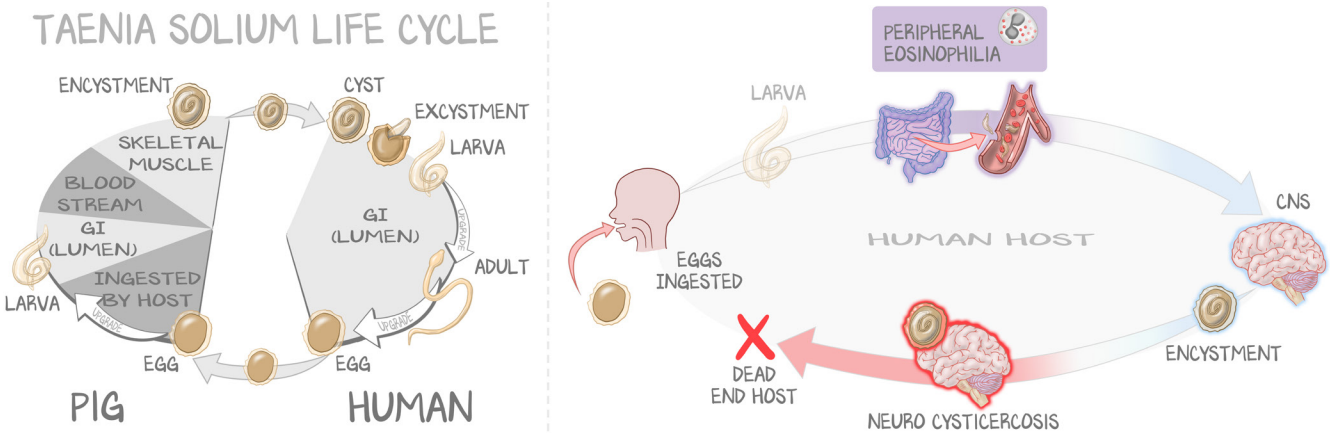

Figure 2.7: *Taenia solium*
What is supposed to happen is on the left. The human has an adult tapeworm in their intestine. They poop out eggs. Other animals eat the eggs, which turn into larvae, penetrate the GI mucosa, and form cysticerci in tissue. The human eats that other animal with the cysticercoid larvae, who upgrade into an adult tapeworm. BUT! If things go out of order and the human eats the egg instead, the egg-taenia can't tell the difference, and this is what causes disease. Start with the human head and follow the arrows with the text. The human swallows eggs, where the eggs upgrade to larvae in the gut. The larvae penetrate the GI mucosa and enter the bloodstream, provoking peripheral eosinophilia. They swim to the tissue, where they encyst, forming cysticerci, causing the disease cysticercosis. When those cysticerci are in the brain, it is called neurocysticercosis. Cysts cause damage to the brain, which can present with seizures and focal neurologic deficits. Human brain is not often the meal of the intended definitive host (other humans). Thus, when the human eats the eggs, the human ends up as the intermediate and dead-end host for those larvae. But only when things go out of order. Because if what's supposed to happen does happen—an animal eats the eggs, gets cysticerci, and is eaten by the human—the encysted larvae become adult tapeworms in the human. So humans are supposed to be the definitive host. It's only if the human eats the eggs instead of the encysted larvae that things go awry.

Diphyllobothrium. A human eats a fish that has larvae in it. The fish was not well cooked. UPGRADE. A human now has the **adult worm** in the intestine. Adult worms release eggs into the stool. Stool-with-eggs contaminates water. Fish are in water, who eat eggs. UPGRADE. Larvae in fish. Larvae form cysts in fish. Human eats fish. While super rare in the US, it is a favorite of board examinations because it is the worm that **eats B_{12}**, which can provoke a B_{12}-deficiency, causing a megaloblastic macrocytic anemia. The deficiency happens because there is no B_{12} to absorb because the worm eats it. See eggs in feces, treat with praziquantel.

***Echinococcus* is liver cysts in humans**. Humans are dead-end hosts and are incidental hosts. Dogs do it to humans. Dog has the adult worm in intestines. Dog poops eggs. Animals (sheep) eat poop, ingest eggs, UPGRADE, larvae in sheep. Larvae cause cysts. Dog eats sheep with cysts. UPGRADE. Adult worm in dog. HUMANS eat EGGS in CONTAMINATED WATER or food, UPGRADE. Humans have larvae. Larvae cause cysts in humans, too. Don't worry about what happens when humans eat the sheep. We are so close to the end. It doesn't matter. *Echinococcus* larvae in humans cause **hydatid cysts**. They do that in sheep. They do that in humans. Hydatid cysts are **huge cysts** that have **little cysts inside the big cyst**. The fluid in this cyst is very antigenic. **Poking one**, thereby releasing its contents, can lead to **anaphylactic shock**. The cysts must be surgically removed. The classic presentation is in the **liver**, though lung, brain, and bone are possible. Treat with albendazole.

This is the "E" parasite that forms liver CYSTS that do need to be removed. (*Entamoeba* causes abscesses you DON'T have to remove.)

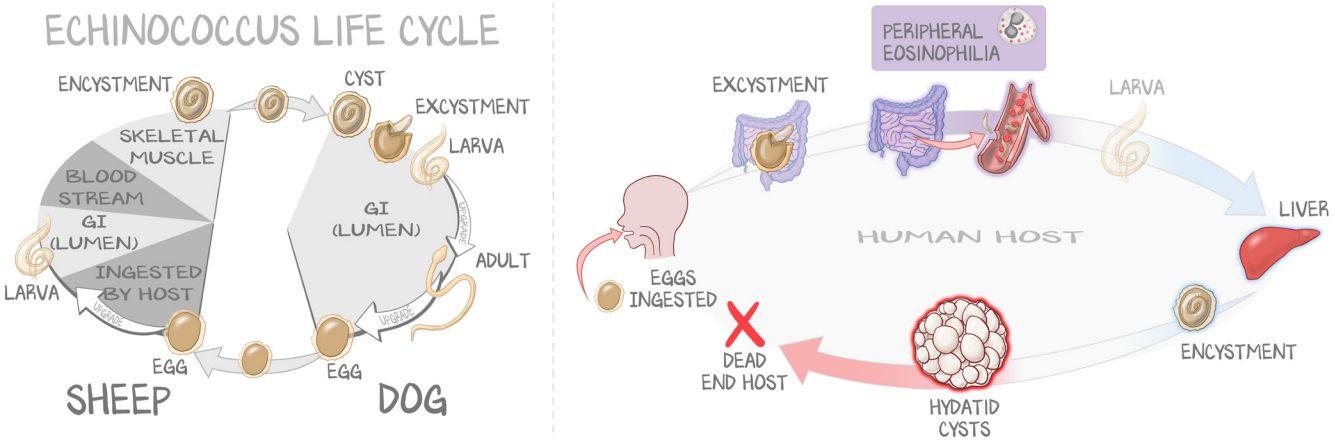

Figure 2.8: *Echinococcus*
Dog definitive host, sheep intermediate host. Everyone remembers the sheep and the dog thing. Ignore the sheep and the dog! Start with the human head and follow the arrows with the text. The human swallows eggs, where the eggs upgrade to larvae in the gut. The larvae penetrate the GI mucosa and enter the bloodstream, provoking peripheral eosinophilia. They swim to the liver, where they encyst. The encystment leads to the formation of hydatid cysts. Human liver is not often the meal of the intended definitive host (even if served with a nice Chianti, Agent Starling). Thus, the human is the intermediate host and the dead-end host.

Trematodes

ZOMFG ALMOST DONE!

Schistosoma causes cirrhosis. *Clonorchis* causes cholangiocarcinoma. Trematodes are worms, but they get a different name, cercariae. We are going to call them worms.

***Schistosoma*.** *Schistosoma* are **free-swimming** worms that **penetrate human skin**. But wait, let's start at the egg form. Eggs enter contaminated water. Eggs become larvae. Larvae infect snails. Snails allow the larvae to undergo asexual reproduction. The larvae leave the snails and find human skin standing in water. Through the skin, the worms get to the blood (cutaneous larvae migrans). *Schistosoma* worms always travel in pairs. In the blood they wait, grow into their adult forms, and reproduce. Their eggs are dropped into the organ to which they are near. Hepatic *Schistosoma*'s eggs end up in the stool. Urinary bladder *Schistosoma*'s eggs end up in the urine. Eggs enter contaminated water.

Hepatic schistosomiasis. *S. mansoni* and *S. japonicum* find their way to the **mesenteric veins**, where their eggs are dropped into the intestine and exit in stool. *S. mansoni* and *S. japonicum* live in the mesenteric veins. They sometimes get washed up to the portal circulation where they lay eggs in the liver. These eggs are fought against with granulomas. This chronic inflammation leads to **cirrhosis**. The fact that the worms are in the blood vessels worsens portal hypertension and accelerates the development of varices.

Urinary bladder schistosomiasis. *S. haematobium* finds its way to the **venous plexus of the urinary bladder**. *S. haematobium* drops eggs into the bladder. Eggs are fought against with granulomas. Chronic inflammation leads to fibrosis and can present with **hematuria**. Chronic inflammation of a squamous epithelium results in **squamous cell carcinoma of the bladder** (most bladder cancers are transitional cell). Treatment is praziquantel.

***Clonorchis*.** Eggs end up in water. Eggs are eaten by snails. UPGRADE. The eggs hatch and become larvae. The larvae leave the snail and infect fish. There, they become encysted larvae within the meat. Humans catch fish with larvae. Humans do not cook fish. Humans eat larvae. UPGRADE. Adults in humans produce eggs. *Clonorchis* goes to the biliary tract and causes **pigmented gallstones** and increases the risk of **cholangiocarcinoma** (gallbladder and biliary tract cancer). Eating raw fish causes gallbladder cancer. Uncommon in the United States but common in island populations in Southeast Asia. Treatment with praziquantel.

WORM	ASSOCIATIONS
Enterobius Pinworm	Live in cecum, where fertilized Female delivers eggs to perianal region Baby picks butt, then puts hands in mouth, inoculating baby Eggs to larvae in intestine, burrow into mucosa to develop Scotch tape test at night Albendazole
Ascaris	Fecal-oral route, eggs to mouth Larvae hatch and burrow through GI mucosa to bloodstream—**peripheral eosinophilia** Exit in the lung—**pulmonary eosinophilia** Human coughs up larvae, which are ingested Larvae mature into adults Does not attach to intestinal wall, but does eat human food **Malnutrition, intestinal obstruction if they get big enough** Albendazole
Ancylostoma, *Necator*	Eggs hatch in soil Larvae in soil, human steps on soil Larvae burrow through the skin to bloodstream—**peripheral eosinophilia, larva migrans** Exit in the lung—**pulmonary eosinophilia** Human coughs up larvae, which are ingested Larvae mature into adults Attach to the intestinal wall, sucking blood, causing **iron def anemia** Albendazole or pyrantel pamoate
Strongyloides	Eggs hatch in human, larvae get into stool Larvae in soil, human steps on soil Larvae burrow through the skin to bloodstream—**peripheral eosinophilia, larva migrans** Exit in lung—**pulmonary eosinophilia** Human coughs up larvae, which are ingested Larvae mature into adults Can also hatch in intestines causing autoinfection—hatch and burrow at once = **Gram-negative bacteremia** Albendazole or ivermectin
Trichinella spiralis	Cysts in skeletal muscle Encysted larvae hang out in skeletal muscle Undercooked meat (**pork**) is consumed, encysted larvae ingested excyst Larvae burrow through GI mucosa into bloodstream—**peripheral eosinophilia** Exit in **skeletal muscle** where they encyst again = **myopathy** Humans dead-end host Albendazole and steroids
Toxocara canis	Eggs are passed in dog feces Human gets dog feces in mouth, eggs are ingested, upgrade to larvae Larvae burrow through GI mucosa into bloodstream—**peripheral eosinophilia** Exit **everywhere**—brain (seizure) heart (myocarditis), eyes (blindness) Humans dead-end host Albendazole

Table 2.3: Nematodes That Use the Human Gut

WORM	ASSOCIATIONS
Onchocerca	Insect vector is blackfly Bite = dermal nodules, adult larvae River blindness—**microfilariae accumulate in the eye** Ivermectin
Loa loa	Insect vector is deer fly, horse fly, mango fly Adult worm crawls across the eye Diethylcarbamazine
Wuchereria bancrofti	Insect vector mosquito (microfilariae only) Microfilariae circulate at night, visualized on **blood smear** of blood taken **at night** Diethylcarbamazine Worms in lymph nodes = **elephantiasis**

Table 2.4: Nematodes Transferred by Insect Vectors

WORM	ASSOCIATIONS
Taenia solium	Humans host adult tapeworms in gut Eggs passed in stool **Livestock eat eggs**, eggs turn into larvae in gut, larvae go to muscle = **cysticerci** Humans eat livestock, undercooked, larvae ingested Larvae become adults and live in human gut, steal food **Humans eat eggs**, eggs turn into larvae in gut, larvae go everywhere = **cysticerci** **Neurocysticercosis** = holes in brain from worm forming cysticerci in brain Praziquantel for not neuro Albendazole for neuro
Diphyllobothrium	Fish eat eggs, eggs turn into larvae, larvae go to fish muscle Human eats fish, undercooked, larvae mature in gut to adults, lay eggs Adults eat B_{12}, resulting in **megaloblastic anemia** and **malabsorption** Praziquantel
Echinococcus granulosus	Human is dead-end host Dog poops eggs, eaten by sheep, eggs become larvae, larvae go to liver Dog eats sheep, eaten larvae mature into adults in dog gut, lay more eggs **Human drinks contaminated water that dog pooped in** and the larvae do the same thing as in sheep, form **liver cysts** called **hydatid cysts** Do not poke cysts, will cause anaphylaxis Must be surgically drained Albendazole

Table 2.5: Cestodes (Tapeworms)

WORM	ASSOCIATIONS
Schistosoma	Humans contaminate water with eggs Eggs hatch in water, larvae (called cercariae) infect snails Larvae leave snails in pairs, burrow through skin of human standing in water **Hepatic** disease = cirrhosis, drop eggs into stool (*S. mansoni, S. japonicum*) **Bladder** disease = squamous cell cancer, drop eggs into urine (*S. haematobium*) Praziquantel
Clonorchis sinensis	Humans contaminate water with eggs Eggs are eaten by snails, which hatch to become larvae in snails Larvae leave snail, infect fish Human eats undercooked fish, larvae mature to adults in human gut Adults live in biliary tree causing **pigmented gallstones** and **cholangiocarcinoma** Praziquantel

Table 2.6: Trematodes (Flukes)